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Review

# The crucifixion of Jesus: Review of hypothesized mechanisms of death and implications of shock and trauma-induced coagulopathy

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ABSTRACT

The crucifixion of Jesus is arguably the most well-known and controversial execution in history. Christian faithful, dating back to the time of Jesus, have believed that Jesus was executed by crucifixion and later returned physically to life again. Others have questioned whether Jesus actually died by crucifixion, at all. From review of medical literature, physicians have failed to agree on a specific mechanism of Jesus' death. A search of Medline/Pubmed was completed with respect to crucifixion, related topics, and proposed mechanisms of Jesus' death. Several hypotheses for the mechanism of Jesus' death have been presented in medical literature, including 1) Pulmonary embolism 2) Cardiac rupture 3) Suspension trauma 4) Asphyxiation 5) Fatal stab wound, and 6) Shock. Each proposed mechanism of Jesus' death will be reviewed. The events of Jesus' execution are described, as they are pertinent to development of shock. Traumatic shock complicated by trauma-induced coagulopathy is proposed as a contributing factor, and possibly the primary mechanism, of Jesus' death by crucifixion.



## 1. Introduction

The crucifixion of Jesus is the most well-known and controversial execution in history. Jesus' early followers firmly believed he died by crucifixion and returned physically to life afterward. It is a historical fact that many of Jesus' early followers suffered violent deaths for refusing to recant this belief.[1] Christianity rose to a major world religion from this small contingent of early followers.

The mechanism of death in crucifixion is not obvious. Physicians have proposed several hypotheses regarding Jesus' mechanism of death in medical literature. These have included, 1) Pulmonary embolism 2) Cardiac rupture 3) Suspension trauma 4) Asphyxiation 5) Fatal stab wound, and 6) Shock. Some have suggested that Jesus survived crucifixion, a proposition that has become popularly known as the "swoon theory".[2]

The historical account of Jesus' execution is taken from biblical records (the Gospels).[3] It is recognized that the biblical records are articles of faith, and as such are the subject of controversy. However, the biblical descriptions of crucifixion are consistent with historical and archeological information.[4] These descriptions of Jesus' crucifixion are taken to be historically accurate for the purposes of this review.

Review of hypothesized mechanisms of Jesus' death, in light of current medical knowledge, is warranted due to deeply held yet conflicting views surrounding the mechanism of Jesus' death, or whether he actually died by crucifixion at all. The events of Jesus' execution are briefly described, as they could have contributed to the development of shock. An acute trauma-induced coagulopathy in Jesus has not previously been presented in medical literature as a possible contributing factor to his death. Shock, complicated by acute trauma-induced coagulopathy, is proposed as a likely contributing factor, and possibly the primary mechanism of Jesus' death by crucifixion.

## 2. Methods

Comprehensive literature searches were performed in MEDLINE/PubMed from 1950-2010 that focused primarily on the subjects of Jesus and crucifixion. Based upon these results, secondary searches were performed on the subjects of suspension trauma, shock, hematidrosis, and trauma-induced coagulopathy. Citations for additional articles and books were extracted from reference lists of relevant articles, and all formed the basis of this review.

## 3. The twelve hours prior to Jesus crucifixion

The evening prior to his execution, Jesus went to a garden to pray after the Passover meal with his disciples. Believing crucifixion

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was imminent, Jesus was observed sweating "*drops of blood*" (Luke 22:44). Psychogenic hematidrosis is a rare phenomenon predominantly observed prior to execution.[5] He was arrested by religious leaders, convicted that night of a capital offense of blasphemy claiming to be the Hebrew Messiah, a divinely appointed king in Hebrew prophetic literature.[6] As Israel was a client kingdom of the Roman Empire, a death sentence could only be pronounced by Roman civil authorities.[7] Therefore, after beating him, Jesus was taken to Roman authorities (Matthew 27:1–2).

Pontius Pilate, the local Roman Prefect, heard charges against Jesus at daybreak. Recognizing the accusations were motivated by jealousy, Pilate had Jesus beaten rather than summarily executed (John 19:1–5). A "whole company of soldiers" then beat Jesus and caned him, his second beating (Mark 15:16-19). He then received a scourging with a *flagrum* (leather whip with bones or lead balls sewn into the ends), Jesus' third beating.[8]

Roman soldiers were known to have anti-Semitic sentiments.[9,10] This likely heightened the brutality of Jesus' beatings, since he was viewed as a political insurgent claiming to be the King of Israel in defiance of Caesar.

Crucifixion was a typical punishment for political insurgents.[11] Pilate's allegiance to Caesar could have been publicly questioned by Jewish leaders, if he did not crucify Jesus (John 19: 6-12). Pilate famously *washed his hands,* likely invoking Hebrew symbolism to publicly absolve himself of responsibility for killing Jesus (Deuteronomy 21:6–9). In apparent reluctant acquiescence, he then ordered Jesus' crucifixion (Matthew 27:24).

At crucifixion, wrists were typically nailed to the *patibulum* (the short section of the cross). For mass crucifixions, or if the supply of nails were limited, arms were tied. The *patibulum* was then lifted and placed on the *stipes,* (the long section of the cross), which was permanently fixed in the ground. The two sections of the cross were held together by a mortise and tenon joint, forming the T-shaped *tau* cross or *crux commissa*.[12] The crucified victim's feet were then fixed to the *stipes*, typically with nails. Limited archeological information suggests nails were driven through the lateral hind foot, fastening the calcaneus to the *stipes*.[13]

Death by crucifixion was slow, marked by pain, environmental exposure, starvation, dehydration, and probable infection.[2,11,13] Jesus expired at 3:00 p.m., after about 6 h on the cross (Matthew 27:45–50). Death this rapid was atypical. Some historical references describe crucifixion lasting several days.[14]

### 3.1. Proposed causes of death

#### 3.1.1. Pulmonary embolism hypothesis

Pulmonary embolism has been suggested as the mechanism of Jesus' death. Hereditary thrombophilia is associated with Jewish populations (Factor V Leiden). Specifically, there is a higher prevalence in the current Galilean Jewish population. It has been suggested that Jesus had this genetic thrombophilia, which lead to thromboembolism and sudden death from being immobilized on the cross.[15]

However, current Galilean genetic profiles would likely differ with those of two thousand years ago due to Jewish migration through the centuries. Additionally, the time on the cross does not seem adequate to develop deep venous thrombosis.[16] Furthermore, Jesus would have been pushing up with his legs, perhaps to facilitate breathing and relieve discomfort in his arms, and was not immobilized.

#### 3.1.2. The "Broken Heart" hypothesis

Cardiac rupture was originally proposed by Stroud in 1847, and popularized among laity as the notion that Jesus died of a "broken heart".[17,18] The most common cause of cardiac rupture is myocardial infarction, causing rapid death by cardiac tamponade. While myocardial infarction was thought to have lead to the demise of Jesus in the past, it has become a less favored explanation in the light of current understanding of cardiac disease.[17]

Cardiac contusion and rupture from his beatings or from falling *en route* to the execution site has been suggested.[19] However, this would require a specific blunt force trauma to the sternum, likely associated with fractures. Traumatic cardiac rupture by this mechanism generally leads to immediate death.[17]

#### 3.1.3. Suspension trauma hypothesis

Suspension trauma has been suggested as a possible mechanism of Jesus' death.[20,21] With prolonged immobilization, orthostatic intolerance can occur from pooling of blood in the lower extremities.[22] Studies of individuals on a tilt table have shown near 90% of subjects will faint in 1 h or less of suspension.[21,23]

Deaths have been reported from suspension trauma. For example, mountain climbers suspended without prompt rescue have died. Autopsies have shown signs of tissue hypoperfusion, suggesting the mechanism of death being hypovolemic shock. (Deep venous thrombosis or embolic phenomena were not observed in those individuals).[23]

Suspension trauma is not analogous to crucifixion. With crucifixion, the victim's feet were affixed by nails. The legs were not immobilized or unsupported.

#### 3.1.4. Suffocation hypothesis

Asphyxiation as the mechanism of Jesus' death was proposed in the 1920's and 1930's by LaBeck and Hynek and later embraced by others.[17] Hynek described torture of prisoners suspended by the wrists with the feet unsupported (*Anbinden*). Victims appeared to have difficulty breathing within minutes, struggling to pull up with their arms to facilitate expiration. This lead Hynek to suggest that suffocation was the likely mechanism of Jesus' death.[24] Similar torture was observed at the Dachau Concentration Camp in World War II. Death by this mechanism was rapid, occurring in about 3 h. Recorded descriptions suggest that victims were unable to exhale, leading to suffocation.[17]

Breathing would have been difficult on the cross, and impaired respiration may have been a contributing factor in death by crucifixion. Musculature attached to the chest wall, such as serratus anterior muscles, arises from the scapula and inserts on the ribs. The extraction force at each arm from hanging on the cross would restrict chest wall movement in a relative expanded position. The diaphragm would also be at a mechanical disadvantage for respiration, by flattening and restricting its movement. It is thought that the crucified victim would have to pull up with his arms to facilitate paradoxical respiration, with inspiration becoming passive and expiration active.[11]

One criticism of the asphyxiation hypothesis for Jesus' death is that wartime torture examples do not compare with Roman crucifixion. In the wartime torture examples, individuals had the hands directly overhead, and legs were unsupported or restrained. Death was also rapid with these wartime torture victims, typically within 3 h, while the torture of crucifixion often lasted much longer.[17]

Interestingly, volunteer re-enactment studies have been done where individuals were suspended on a cross.[25,26] Respiratory distress was not observed in either study. Hypotension, leg edema, and oxygen desaturation did not occur with these individuals, although Ball noted a decrease in forced inspiratory vital capacity.[25] Such re-enactment studies cannot be considered directly comparable to crucifixion, but they further question whether asphyxiation could have been the primary mechanism of Jesus' death.[13,17]

#### 3.1.5. Fatal stab wound hypothesis

"But when they came to Jesus and found that he was already dead, they did not break his legs. Instead, one of the soldiers pierced

Jesus' side with a spear, bringing a sudden flow of blood and water" (John 19:33–34). The idea that Jesus was alive until a spear impaled his chest is based on the assumption blood cannot flow from a corpse, and presumes that if Jesus were dead, his blood would have been clotted.

A pleural effusion, unless it is loculated, settles according to gravity. In a crucifixion victim, this would be in the anterior inferior aspect of the chest cavity. A pleural effusion could have arisen from heart failure or perhaps from his beatings and blunt trauma to the chest wall. A spear entering the chest would first tap a pleural effusion, if present, having the appearance of water. Next, it would most likely enter the right atrium causing blood to appear.[11,27] This would cause immediate death by cardiac rupture. Inflicting this type of chest stab wound was likely a matter of protocol to assure no crucifixion victim escaped death, particularly if the body were going to be released.[11,28]

The proposition that Jesus' death occurred by a fatal stab wound is possible. This would certainly result in immediate death and would have an appearance consistent with biblical descriptions. However, the *exactor mortis*, a Roman centurion supervising the crucifixion, would have credible expertise in pronouncing death of crucifixion victims. Jesus was determined to be dead, prior to receiving the chest stab wound.

Concluding Jesus was alive from the observation of blood emanating from the stab wound does not consider the possibilities of incomplete clotting, clot liquefaction, purge fluid, or the potential effects of shock on blood coagulation. The observation of blood flow from Jesus' chest wound does not mandate the conclusion that he was alive at that moment.

### 3.1.6. The shock hypothesis

Shock has been proposed as the mechanism of Jesus' death.[11,29] Shock is a state of insufficient perfusion of vital organs with successive imbalance of oxygen supply and demand due to an intravascular volume deficiency with critically impaired cardiac preload. Traumatic hemorrhagic shock is a subtype. Hemorrhage is the most common cause of decreased circulatory volume in trauma.[30,31] Tissue ischemia, systemic inflammation, coagulopathy, and the metabolic effects of shock can lead to multiorgan system failure and cardiovascular collapse.

A number of factors could have contributed to shock in Jesus. First, hematidrosis is indicative of his intense mental anguish during the night prior to his crucifixion. While it is unlikely that Jesus lost a large amount of blood from hematidrosis, it does speak to his mental condition at the time. In such a state of anxiety, it is likely that he was sweating profusely through the night. Second, he was deprived of fluids. Third, there would have been blood loss from Jesus' crown of thorns, his multiple beatings, and his pre-execution scourging. Fourth, he may have developed a pleural effusion which would have caused a fluid shift.

By the time Jesus was finally compelled to carry the *patibulum* (the short section of the cross) 500 m to the crucifixion site, he was unable to do so. The executioners enlisted the help of a bystander to carry it for Jesus (Matthew 27:32). Jesus cried out in thirst on the cross, suggesting dehydration and possible early stage shock (John 19:28).[32]

During shock, decreased blood flow from diminished circulatory volume causes catecholamine, vasopressin, rennin-angiotensin, and cortisol responses which increase heart rate and cause constriction of vascular beds. Tissue swelling from ischemia can worsen hypoperfusion effects by further reducing local tissue blood flow. Pneumothorax, if present, can decrease preload and worsen hypoperfusion as well.[31]

The resulting tissue ischemia causes metabolic acidosis and release of inflammatory mediators having systemic effects. Ischemic tissue produces lactic acid and free radicals, causing local and systemic toxic effects. Inflammatory factors are released. These include tissue necrosis factor, prostocyclin, prostaglandins, leukotrienes, and interlukens, among others. These tissue effects can cause acidemia and systemic inflammation.[31]

Zugibe described a case of "traumatic wet lung" and pneumothorax on autopsy of a subject beaten with an electrical cord.[33] Similar lung trauma could have easily been present in Jesus from his extensive beatings. If present, chest trauma, pneumothorax, pleural effusion, or adult respiratory distress syndrome can diminish oxygen delivery as well as diminish venous return and cardiac preload.[32] (A respiratory mechanism of death cannot be excluded). Respiratory acidosis can add to the metabolic acidosis arising from tissue ischemia in shock.

Acute irreversible traumatic shock can lead to death within hours.[31] Jesus' unusually rapid death on the cross suggests this pathomechanism. Prolonged hypoperfusion eventually leads to endothelial energy depletion, causing progressive vasodilation and vascular non-responsiveness to catecholamines. The effects of sustained tissue ischemia and release of inflammatory mediators, can lead to an irreversible state of shock in spite of the best treatment efforts. If afforded modern resuscitation, the victim may normalize vital signs but later succumb to multiorgan system failure from the metabolic consequences of severe or prolonged ischemia. Coagulopathy, capillary leakage, vasodilation, and hypovolemia can lead to cardiopulmonary compromise and cause rapid death.[31] Progressive shock can lead to rapid death by effective exsanguination.

### 3.1.7. Trauma-induced coagulopathy

A recognized complication of traumatic shock is trauma-induced coagulopathy. This is said to occur in approximately 25% of trauma patients.[34] This acute coagulopathy begins early in the post-injury period. It is an independent prognostic indicator of poor outcome, having four times the likelihood of fatality when present.[35]

Trauma-induced coagulopathy is likely to occur when several factors are present simultaneously. Specifically, these are shock, tissue injury, hypothermia, acidemia, and systemic inflammation.[34] Coagulopathy, hypothermia, and acidemia have been called the "lethal triad" and can lead to progressive derangement of clotting mechanisms.[36] When the components of the lethal triad occur together, the deleterious effects are compounded. This "lethal triad" has been associated with mortality rates nearing 60%. Current treatment efforts include thermoregulation, control of bleeding, careful resuscitation, replenishment of blood products, and pH monitoring.[37] In the best of trauma centers this can be difficult to manage, but in Jesus' time this acute coagulopathy would lead to certain rapid death.

Jesus had extensive tissue damage from multiple beatings plus widespread lacerations on the trunk and extremities from scourging. Diffuse tissue damage and hemorrhage can lead to depletion of platelets and coagulation factors. The potential loss of platelets and clotting factors from bleeding and diffuse injury cannot be underestimated. This could cause a consumptive component of coagulopathy further contributing to derangement of clotting mechanisms. The extent of tissue damage appears to directly correlate to the development and severity of trauma-induced coagulopathy.[34,37] While trauma-induced coagulopathy is considered a separate entity from consumptive coagulopathies, platelet and clotting factor depletion could potentiate an acute coagulopathy associated with trauma.

Jesus certainly could have been hypothermic from vasoconstriction in his extremities, blood loss, profuse sweating from anxiety, and environmental exposure hanging naked on the cross. (Ambient average temperatures during the first week of April in

Jerusalem range from 8 to 14° Celsius, www.weather.com). The multiple enzymatic reactions associated with the clotting mechanism function best at approximately 37° Celsius. Hypothermia alone will impair blood clotting, particularly at temperatures less than 35 C, as it slows enzymatic activity.[34] Hypothermia also impairs platelet function and plug formation by decreasing Von Willebrand Factor interaction with platelet glyoproteins, ultimately leading to decreased thrombin generation.[36,37] At the same time, hypothermia activates fibrinolysis and clot lysis.[37] These effects are a dangerous combination.

Metabolic acidosis occurs secondary to tissue ischemia during shock. Reduction in pH has progressive effects of decreasing plasma protease activity, particularly at a pH of 7.35 or less.[37] Presumably, the increased hydrogen ion concentration inhibits the interaction between clotting factors and the negatively charged phospholipids on the cell wall of activated platelets.[36] Acidosis can increase clotting time and also decrease clot strength.[34] The effects of pH reduction in decreasing enzymatic activity and thrombin generation are compounded when hypothermia is also present, greater than the sum of expected effects from acidosis or hypothermia separately.[36,37]

Trauma-induced coagulopathy may have been a contributing factor, if not the primary factor, in Jesus' death. It would explain how Jesus' death could occur so rapidly, namely 6 h, rather than several days. It would also explain how blood could flow from Jesus' corpse when his chest was impaled by the spear.

## 4. Conclusion

The specific mechanism of Jesus' death, or combination of contributing factors, cannot be proven. That Jesus suffered a brutal death by torture and crucifixion seems clear. Shock, complicated by trauma-induced coagulopathy, may have been a contributing factor and could possibly have been the primary mechanism of Jesus' death.

*Conflict of interest*

The authors have no conflict of interest to declare.

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